J. Helminthol. Soc. Wash.
58(1), 1991, pp. 152–155

Research Note

A Survey for Trichinosis in Selected Predatory and Scavenger Birds in Montana, with an Evaluation of the Infectivity of Two Mammalian *Trichinella spiralis* Isolates in Birds

DAVID E. WORLEY, ROLANDO H. ESPINOSA, AND FLOYD M. SEESEE
Veterinary Molecular Biology Laboratory, Montana State University, Bozeman, Montana 59717

ABSTRACT: Muscle samples from 103 free-ranging predatory and scavenger birds of 27 species from western Montana were digested and examined for *Trichinella* larvae during the period from 1971 to 1988. Families represented included accipiters, falcons, and strigids; golden and bald eages, red-tailed and Cooper's hawks, and great horned owls predominated. Additional specimens of fish- or carrion-feeders such as herons, loons, ospreys, crows, ravens, and turkey vultures also were examined. No infected individuals were found. Attempts to induce experimental infections in a short-eared owl, long-eared owl, and crow with *T. spiralis* isolates from a grizzly bear and fisher also were unsuccessful. The apparent absence of avian trichinosis in the wild bird population and the inability to induce experimental infections in captive birds suggest that birds play little or no obvious role in the epizootiology of sylvatic trichinosis in the high plains/Rocky Mountain region of western Montana.

KEY WORDS: *Trichinella spiralis,* avian infections, cross-transmission.

Although *Trichinella spiralis* (Owen, 1835) probably has the broadest host range of any helminth (Gould, 1970; Despommier, 1983), under natural conditions the various *T. spiralis* subspecies are thought to be infective only in mammals (Gould, 1970; Despommier, 1983). The related species *T. pseudospiralis,* originally described from the raccoon *Procyon lotor* in Dagestan, Northern Caucasus (Garkavi, 1972), is believed to be a normal parasite of birds (Miroshnichenko, 1976; Shaikenov, 1980), although under experimental conditions mice and other rodents are susceptible (Tomašovičová, 1975). Subsequent avian records of *T. pseudospiralis* have extended the known distribution of the species to Spain (Calero et al., 1978) and the United States (Wheeldon et al., 1983).

Although the natural occurrence of *T. pseudospiralis* has been demonstrated on 3 continents, its distribution and the species involved in transmission are virtually unknown. The primary purpose of the present study was to screen tissues from a series of raptorial and scavenger birds for the presence of *Trichinella* larvae in a region of high trichinosis endemicity in mammals (Worley et al., 1974, 1982). A secondary objective was to evaluate the infectivity of 2 mammalian *T. spiralis* isolates in birds likely to be exposed to this infection via their normal feeding behavior as predators or scavengers on known mammalian reservoirs of *T. spiralis* in western Montana, northern Wyoming, and eastern Idaho.

Samples of breast, thigh, or other muscle were obtained from eagles, hawks, owls, and other raptors submitted to the Raptor Rehabilitation Program at Montana State University but found unsuitable for release. Other avian specimens were provided by the Montana Department of Fish, Wildlife and Parks or were obtained from road kills. The classification used was that of the A.O.U. Check-list of North American Birds, 6th ed. One 25-g muscle sample was examined in most instances. Occasionally, tissues from 2 sites were screened for larvae.

Tissues were removed from some birds shortly after death. In most instances, carcasses were frozen for several months before muscle samples were removed and processed. Tissues were cut into small pieces with scissors prior to comminuting with an Omnimixer. Homogenized tissue then was digested in 1% pepsin–0.7% HCl for 5–12 hr at 37°C under constant agitation. The digested material was washed on a 200-mesh screen to remove soluble debris prior to examination of the retained material for *Trichinella* larvae with a dissecting microscope.

The infectivity of 2 mammalian *T. spiralis* isolates in birds was tested by oral inoculation of 2 owls and a crow with larvae originally isolated from a grizzly bear (*Ursus arctos*) and a fisher (*Martes pennanti*) in western Montana. Both biotypes were maintained in laboratory-reared deer mice (*Peromyscus maniculatus*) at the Montana State University Animal Resources Center. Larval suspensions obtained by peptic digestion of skinned, eviscerated mouse carcasses were ad-

Table 1. Wild birds negative for *Trichinella spiralis* larval tissue infections in western Montana (1971–1988).

Family	Species	No. examined
Gaviidae	Common loon (*Gavia immer*)	1
Ardeidae	Great blue heron (*Ardea herodias*)	1
Cygnidae	Trumpeter swan (*Olor buccinator*)	1
Cathartidae	Turkey vulture (*Cathartes aura*)	1
Accipitridae	Goshawk (*Accipiter gentilis*)	1
	Sharp-shinned hawk (*Accipiter striatus*)	1
	Cooper's hawk (*Accipiter cooperii*)	6
	Red-tailed hawk (*Buteo jamaicensis*)	7
	Rough-legged hawk (*Buteo lagopus*)	2
	Ferruginous hawk (*Buteo regalis*)	1
	Golden eagle (*Aquila chrysaetos*)	17
	Bald eagle (*Haliaeetus leucocephalus*)	5
	Marsh hawk (*Circus cyaneus*)	3
Pandionidae	Osprey (*Pandion haliaetus*)	1
Falconidae	Gyrfalcon (*Falco rusticolus*)	1
	Prairie falcon (*Falco mexicanus*)	3
	Peregrine falcon (*Falco peregrinus*)	2
	American kestrel (*Falco sparverius*)	4
Strigidae	Great horned owl (*Bubo virginianus*)	34
	Great gray owl (*Strix nebulosa*)	3
	Long-eared owl (*Asio otus*)	2
	Short-eared owl (*Asio flammeus*)	1
	Saw-whet owl (*Aegolius acadicus*)	1
Caprimulgidae	Common nighthawk (*Chordeiles minor*)	1
Picidae	Northern flicker (*Colaptes auratus*)	1
Corvidae	Common raven (*Corvus corax*)	1
	Common crow (*Corvus brachyrhynchos*)	1

ministered via stomach tube in physiological saline solution. Recipient birds were maintained in individual cages for 26–45 days postinoculation prior to euthanasia. At necropsy, tissue samples were collected from wing, thigh, gastrocnemius, and pectoralis muscle, when available, for evaluation for larvae.

A total of 103 birds was examined for *Trichinella* muscle larvae between 1971 and 1988 (Table 1). Included in this series were 27 species representing 11 families: 9 accipiters, 4 falcons, and 5 owls. Additional fish or carrion feeders such as herons, loons, ospreys, crows, ravens, and vultures were examined when available. A few insectivorous or planktonic feeders also were included, i.e., trumpeter swan, nighthawk, and flicker on the basis of possible involvement of insects or other invertebrates in transmission of *T. spiralis*.

No evidence of *Trichinella* muscle larvae was seen in any of the material examined. Because of the limited availability of most species, the status of avian trichinosis in western Montana remains unclear. However, the absence of infection in 65 individuals of 5 major raptorial species (golden and bald eagle, red-tailed hawk, Cooper's hawk, and great horned owl) suggests that these birds appear to play no demonstrable role in biological transmission of *Trichinella* sp. in a region where this parasite occurs widely as a sylvatic infection in mammalian hosts (Worley et al., 1974; Worley and Greer, 1982). The absence of infection in the Cooper's hawk is pertinent also in view of the first North American report of *T. pseudospiralis* in this species in California (Wheeldon et al., 1983). Further, the absence of the parasite in species such as the great horned owl, *Bubo virginianus,* which is known to feed on the common skunk, *Mephitis mephitis* (Bent, 1938), a known host of *Trichinella* in western Montana (Worley and Greer, 1982), suggests that skunk-to-owl transmission may be unlikely to occur as a result of natural exposure via predation or scavenger feeding behavior. On the other hand, Zimmermann and Hubbard (1963) found that trichinosis occurred as a low level infection in the great horned owl in Iowa.

Limited experimental evidence from the present study confirmed the inability of 2 mammalian isolates to develop in birds (Table 2). No

Table 2. Experimental inoculation of birds with mammalian *Trichinella spiralis* larvae.

Host	Larval dose	Source of isolate	Muscles examined postmortem	Tissue larvae recovered*
Short-eared owl (*Asio flammeus*)	865	fisher (*Martes pennanti*)	wing	0
			pectoralis	0
			thigh	0
			gastrocnemius	0
Long-eared owl (*Asio otus*)	8,100	grizzly bear (*Ursus arctos*)	wing	0
			pectoralis	0
			thigh	0
			gastrocnemius	0
Crow (*Corvus brachyrhynchos*)	10,000	fisher (*Martes pennanti*)	wing	0
			thigh	0
			gastrocnemius	0

* Via peptic digestion of muscle samples 26–45 days postinoculation.

tissue invasive stages were found in either of 2 owls or 1 crow given single oral doses of 865–10,000 larvae. The inability to induce infections that progressed past the enteral stage in birds given *T. spiralis* isolates from mammals is consistent with most previous observations. Doerr and Schmidt (1930) attempted unsuccessfully to transmit *T. spiralis* from rodents to hens. Augustine (1933) found that some *Trichinella* larvae given to baby chicks survived in the intestinal tract to produce migratory tissue larvae, but they died in the musculature within a short time. Matoff (1938) reported that pigeons were also an unsuitable host for rodent-derived *Trichinella.* On the other hand, Nemeseri (1968) found that by lowering the natural resistance of chickens by starvation or by feeding an inadequate diet, it was possible to induce disseminated tissue infections, and these larvae were infective to white rats. Overall, the preponderance of experimental evidence suggests that *T. spiralis* isolates derived from pigs or rodents are not infective to galliform or columbiform birds.

Results of the present experimental study complement these findings by demonstrating that some strigiform and passeriform species also are refractory to infection with *T. spiralis* isolates from wild carnivores. The possibility that raptors such as eagles and certain hawks that travel extensively during seasonal migrations could transport muscle larvae mechanically to establish new sylvatic foci is an additional consideration that was outside the scope of the present study. Hence, the role of birds in the epizootiology of trichinosis in the western U.S.A. may be associated with their ability to disseminate infective larvae of mammalian *Trichinella* biotypes rather than as biological reservoirs in the traditional predator/scavenger–prey cycle.

We acknowledge the assistance of Mauritz Sterner and Dan Palmisciano in obtaining and processing tissue specimens for the study. Contribution no. J-2318, Veterinary Molecular Biology Laboratory, Montana State University, Bozeman, Montana.

Literature Cited

Augustine, D. L. 1933. Experimental trichinosis in chicks. Science 78:608–609.

Bent, A. C. 1938. Life histories of North American birds. Pages 306–307 *in* U.S. National Museum, Smithsonian Institution Bulletin 170.

Calero, R., F. Martinez, S. Hernandez, and I. Acosta. 1978. Parasitacion de *Buteo buteo* (Aves: Accipitridae) por *Trichinella* sp. en el Parque de Jerez de la Frontera. Revista Iberica de Parasitologia 38: 135–138.

Despommier, D. D. 1983. Biology. Pages 75–142 *in* W. C. Campbell, ed. *Trichinella* and Trichinosis. Plenum Press, New York.

Doerr, R., and G. W. Schmidt. 1930. Experimentelle Beinflussung der Trichinenwanderung. Die natürliche Immunität des Huhnes. Zentralblatt für Bakteriologie. I. Originale 115:427–430.

Garkavi, B. L. 1972. [*Trichinella* species from *Procyon lotor.*] Pages 53–54 *in* Materials of the All-Union Conference on the Problems of Trichinellosis in Man and Animals, 30 May–1 June. Akademiya Nauk Litovskoi SSR, Vilnius.

Gould, S. E. 1970. Trichinosis in Man and Animals. Charles C Thomas, Springfield, Illinois. 540 pp.

Matoff, K. 1938. Zür Frage der Muskeltrichinellose beim Geflügel. Zeitschrift für Infektionskrankheiten Haustiere 54:116–134.

Miroshnichenko, L. S. 1976. [The susceptibility of birds to trichinelliasis.] Pages 86–90 *in* Materialy dokladov k II-ol zhivotnykh (27–28 Maya 1976).

Akademiya Nauk Litovskoi SSR, Institut Zoologii i Parazitologii, Vilnius, USSR.

Nemeseri, L. 1968. Invasionversuche beim Geflügel mit *Trichinella spiralis.* Helminthologia 8/9:417–420.

Shaikenov, B. 1980. Spontaneous infection of birds with *Trichinella pseudospiralis* Garkavi, 1972. Folia Parasitologica 27:227–230.

Tomašovičová, O. 1975. Poultry—a new host of *Trichinella pseudospiralis* (Garkavi, 1972). Biologia (Bratislava) 30:821–826.

Wheeldon, E. B., T. A. Dick, and T. A. Schulz. 1983. First report of *Trichinella spiralis* var. *pseudospiralis* in North America. Journal of Parasitology 69:781–782.

Worley, D. E., J. C. Fox, J. B. Winters, and K. R. Greer. 1974. Prevalence and distribution of *Trichinella spiralis* in carnivorous mammals in the United States northern Rocky Mountain region. Pages 597–602 *in* Proceedings of the Third International Conference on Trichinellosis.

———, **and K. R. Greer.** 1982. Trichinosis in wild carnivores. Pages 224–227 *in* E. T. Thorne, ed. Disease of Wildlife in Wyoming. Bulletin 9, Wyoming Game and Fish Commission, Cheyenne.

Zimmermann, W. J., and E. D. Hubbard. 1963. Wildlife reservoirs of *Trichinella spiralis.* Pages 194–199 *in* Proceedings 100th Annual Meeting American Veterinary Medical Association.

IN MEMORIAM

J. ALLEN SCOTT
1898–1990

James Allen Scott is among the last of a group of distinguished parasitologists who studied under Hegner, Stoll, Root, and Cort of the Johns Hopkins University School of Hygiene and Public Health in the first quarter of this century. He died at home after an extended illness on 18 August 1990.

Scotty was born in Binghamton, New York, on 2 June 1898. He attended high school in Binghamton and after graduation spent 2 years at Wyoming Seminary in Kingston, Pennsylvania. He spent a short time in the U.S. Army as a Chief Bugler (he enjoyed telling of his experiences as a bugler) and then enrolled in Wesleyan University, Middleton, Connecticut, and graduated with a B.A. in 1922 and an M.A. in 1924. He taught for a brief time at the University of Vermont before entering Johns Hopkins University in 1925. He received his Sc.D. in 1927 under the tutelage of W. W. Cort and carried out monumental studies on hookworm in dogs and cats. He remained at Johns Hopkins as an instructor and research associate in helminthology, and in 1929 he became a member of the field staff in the International Health Division of the Rockefeller Foundation and carried out field research in Egypt. He worked in Egypt until 1936 and carried out extensive studies on hookworm and schistosomiasis. Upon his return from Egypt, he spent a year at Johns Hopkins writing up his research. His work in Egypt has become classic and is often referred to today in that country, especially his comprehensive studies on schistosomiasis. In 1937 he was called again to an overseas assignment where he worked on schistosomiasis in Venezuela until 1940. Again he returned to Johns Hopkins to write up his work. From 1941 to 1944 he was a Visiting Professor at Ohio State University, worked on malaria and hookworm in Georgia, and then put his statistical expertise to use for awhile at the U.S. Census Bureau.

In 1944 Scotty joined the faculty at the University of Texas Medical Branch in Galveston, Texas, where he became a Professor of Statistics and Epidemiology in the Department of Preventive Medicine. He established the Laboratory of Helminth Research and carried out continuous studies on the cotton rat filaria *Litomosoides carinii.* These studies were some of the earliest to be done on filarial immunity. He established a graduate program in helminthology, and among his students were Leroy J. Olson, N. Ted Briggs, and John H. Cross. A number of post-doctoral students also had the opportunity to work with Scotty: Etta Mae MacDonald, Haig Najarian, Ahmed Zien Eldin, among the many.

In 1962 and after the publication of over 100 papers, Scotty left academics and bench research to join the staff of the National Institutes of Health. He became a Health Science Administrator and Chief of the Special Research Grants